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1	DRAFT BORON TOXICITY PROFILE

BY: BILL STONE **DATE:** 5/10/91

VIA: ☐ First Class ☒ Federal Express ☐ Messenger ☐ UPS

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BORON TOXICITY PROFILE

A. Introduction

The purpose of this toxicity profile is to provide currently available information on the health and environmental data concerning boron, in advance of the completion of the Baseline Risk Assessment. Boron is a naturally occurring element essential for plant growth. Some findings suggest that boron is an essential human nutrient and that boron supplements in the diet may help to prevent osteoporosis. In the various studies that have evaluated whether boron is a carcinogen or a mutagen, no evidence of carcinogenicity or mutagenicity has been found.

Some studies involving exposure to high levels of boron have reported certain effects in humans. As explained in detail below, data suggesting human health effects from boron ingestion are limited and based on specific case studies. Similarly, animal studies involving exposure to high levels of boron have also reported certain effects.

In evaluating the significance of any toxicological study, it must be kept in mind that many studies suffer from lack of adequate control for variables, small sample sizes, short-term duration, and lack of statistically significant results. Further, the studies do not always establish a cause and effect relationship between the chemical and the toxicological endpoint.

B. Background

Boron, a nonmetallic element, is ubiquitous in the environment (Minoia et al. 1987). Boron is an essential element in plants and is therefore distributed widely in foods of plant origin. Boron is also an essential nutrient in animals, with a role in mineral metabolism, making it important for maintaining healthy bones. Findings of recent studies suggest that boron supplements in the diet may help to prevent calcium loss and bone demineralization in post-menopausal women, and thus may be an important nutritional factor in determining the incidence of osteoporosis (Nielsen 1988a). Estimates of dietary intake range from 1.7 to 7 mg of boron per day (Nielsen 1988b).

The most common commercial compounds of boron are boric acid and borax (Sprague 1972). Boron compounds are used in a wide range of products, including glassware, soaps, cleansers, water softeners, enamels and glazes. Since boron is an essential trace element for plant growth, borax is frequently applied in fertilizers (Butterwick et al. 1989; Sprague 1972). Borates are also used in insecticides and timber preservatives, and as algicides in water treatment (Procter & Gamble 1987).

Boron is not found in its elemental form in nature; it is usually found in the form of borax, borates, boric acid, and certain borosilicate minerals (USEPA 1976; Carriker et al. 1976). The average concentration of total boron in the earth's crust is about 10 parts per million (ppm), with normal soils ranging from 2 to 100 ppm (Adriano 1986). The concentration of boron in sea water has been reported to be approximately 4.5 mg/liter (mg/L) (U.S. Fish and Wildlife Service 1990). Naturally elevated boron levels are usually associated with marine sediments, large deposits of boron minerals and certain ground waters. Human activities have resulted in elevated boron concentrations in mine drainage and agricultural drainage waters (U.S. Fish and Wildlife Service 1990). Concentrations of boron in fresh waters of the United States average 0.1 mg/L, although levels in western arid regions may range up to 15 mg/L (U.S. Fish and Wildlife Service 1990). The concentration of boron in well water is more variable than that in surface water; concentrations of boron in well water often greatly exceed the average found in surface water (Bingham 1973). The predominant species of boron in most fresh water environments (pH 6-9) is boric acid, regardless of its initial formulation (Butterwick et al. 1989).

This toxicity profile will focus primarily on boric acid, because it is the predominant species in most fresh water environments, and borax since it is commonly found in nature and is frequently applied in fertilizers.

C. Summary of Toxicity

1. Mammalian Toxicity

The toxicity of boron varies significantly depending on the particular boron compound. The boron compounds focused on in this toxicity profile, borax and boric acid, have relatively low acute toxicity for mammals (Sprague 1972). There is no evidence that boron is a carcinogen. Animal studies have shown no increased tumor rate at any level of boron tested. Signs and symptoms of acute boron toxicity in laboratory animals exposed to high levels of boron include depression, lethargy, ataxia, convulsions, discoloration of the spleen, liver and renal medullae, and hyperplasia and dysplasia of the forestomach (Weir and Fisher 1972; NTP 1987). One study claimed that exposure to boron oxide dust may cause conjunctivitis and erythema (Wilding et al. 1959). Weir and Fisher (1972) reported oral median lethal doses (LD_{50}s) in rats ranging from 510-690 mg boron/kg for borax, and 550-710 mg boron/kg for boric acid.

The lethal dose of boric acid and borax is not clearly established. One author has reported the lethal dose to be approximately 2-3 grams for infants, 5-6 grams for children and 15-20 grams for adults (Larsen 1988). Systemic toxicity has resulted from ingestion of 0.17 to 0.2 grams boric acid/kg body weight (Schillinger et al. 1982).

Organs most severely affected by boric acid and borates include the gastrointestinal tract, central nervous system, skin, liver and kidneys.

As discussed below, animal studies have suggested that boron may cause reproductive toxicity after subchronic or chronic exposure. Various studies have reported testicular atrophy, impaired spermatogenesis, reduced testicular and epididymal weights, germinal aplasia, and sterility in animals fed boric acid or borax.

The USEPA (1990) has recommended an oral Reference Dose (RfD) for boron of 0.09 mg/kg/day. After adjusting this value for daily dietary intake of boron (discussed in detail in Section E of this report), an acceptable level of 1.05 mg/L in drinking water can be calculated (Table 1). The USEPA Office of Water has also issued a draft Lifetime Health Advisory for boron of 0.63 mg/L which the USEPA has rounded to 1 mg/L. These values are in agreement with permissible criteria reported by the U.S. Department of the Interior (1968) for raw surface water (1 mg/L) and criteria proposed by the U.S. Fish and Wildlife Service (1990) for the protection of human health (1.0 mg/L). Thus, the USEPA, the U.S. Department of the Interior, and the U.S. Fish and Wildlife Service agree that long-term exposure to boron in drinking water at levels up to 1 ppm is protective of human health.

TABLE 1
Summary of Boron Criteria Recommended by the U.S. Fish and Wildlife Service for the Protection of Public Health and the Environment

	Criterion (mg/L)
Drinking water	1.0
Crop irrigation water	0.3
Aquatic life	1.0
Livestock drinking water	5.0

2. Environmental Toxicity

Boron is an essential nutrient for the growth of higher plants, but above certain threshold concentrations can be phytotoxic. Plant species especially sensitive to the effects of boron include citrus fruits, stone fruits and nut trees. The aquatic species most sensitive to the effects of boron is the rainbow trout in its early life stages. A lowest-observable-effect concentration (LOEC) of 0.1 mg boron/L was reported in reconstituted water; an LOEC of 1.0 mg boron/L was recorded under natural water exposures. Current boron criteria recommended by the U.S. Fish and Wildlife Service for the protection of sensitive species include 0.3 mg/L in crop irrigation waters, 1.0

mg/L for aquatic life, and 5.0 mg/L in livestock drinking waters (U.S. Fish and Wildlife Service 1990) (Table 1).

D. Mammalian Toxicity

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1. Acute Toxicity

a) Animal Data

The toxicity of boron varies significantly depending on the particular boron compound. Borax and boric acid have relatively low toxicity for mammals (Sprague 1972). No deaths were reported in dogs administered up to 738 mg boron/kg as borax or up to 697 mg boron/kg as boric acid via the oral route, although vomiting did occur within one hour of administration (Weir and Fisher 1972). Oral LD_{50}s (in mg boron/kg) of boric acid and borax were reported as 898 and 642 mg/kg, respectively, in rats by Smyth et al. (1969). Weir and Fisher (1972) reported oral LD_{50}s in rats ranging from 510-690 mg boron/kg for borax, and 550-710 mg boron/kg for boric acid. The signs of toxicity for both boric acid and borax included depression, ataxia and convulsions. In a 14-day study, 5 mice per sex were fed diets containing 0, 6200, 12,500, 25,000, 50,000 or 100,000 ppm boric acid. All males fed up to 12,500 ppm and all females fed up to 50,000 ppm survived treatment. Eighty percent of males receiving 25,000 ppm and 60% of males receiving 50,000 ppm survived the treatment. None of the males receiving 100,000 ppm survived. Eighty percent of females receiving 100,000 ppm also survived the treatment. The mice receiving 25,000 ppm or greater exhibited lethargy, decreased body weights, discoloration of the spleen, liver and renal medullae, and hyperplasia and dysplasia of the forestomach (NTP 1987). Verbitskaya (1975) reported oral LD_{50}s in guinea pigs, rabbits and dogs of 332.5 and 306 mg boron/kg, for boric acid and borax, respectively. Direct exposure to large amounts of boron oxide dust reportedly produced conjunctivitis and erythema in rabbits (Wilding et al. 1959).

b) Human Data

The minimum lethal dose of boric acid or borax for man is not clearly established. Larsen (1988) reported single lethal oral doses of boric acid and borax as 2-3 grams for infants, 5-6 grams for children and 15-20 grams for adults. Clayton and Clayton (1981) reported a somewhat higher lowest oral lethal dose of boric acid for man, 640 mg/kg. Systemic toxicity has resulted from ingestion of 0.17 to 0.2 g boric acid/kg body weight (Schillinger et al. 1982).

Boric acid poisoning has reportedly resulted from oral intake by infants or from skin absorption after treatment for infections. Hallet (1955) reported that baby powder containing boric acid applied to the injured skin of an infant for several days caused the development of "blisters" around the buttocks and upper thighs; the infant died after a few days. Infants who ingested from 4.51 to 14 grams of boric acid in formula died within 3 days after exposure (Wong et al. 1964).

Organs affected by boric acid and borax include the gastrointestinal tract, central nervous system, skin, liver and kidneys. Central nervous system symptoms include headache, tremors, restlessness, convulsions, lethargy, delirium, coma and degenerative changes in the brain. Gastrointestinal symptoms include nausea, vomiting, diarrhea and abdominal cramps. Exfoliative dermatitis characterized by a "scalded skin" or "boiled lobster" appearance and desquamation are reported dermatological symptoms. Other reported symptoms of boric acid poisoning include erythema, hypothermy, weariness, circulatory collapse, tachycardia, cyanosis, and degenerative changes in the liver and kidney (USEPA 1975; Clayton and Clayton 1981; Larsen 1988; Locatelli et al. 1987; FDA 1980).

2. Chronic (and Subchronic) Toxicity

a) Animal Data

In a 13-week National Toxicology Program (NTP) oral study, mice were fed 0, 35, 72, 144, 288 or 577 mg boron/kg/day as boric acid. All male mice fed up to 144 mg boron/kg/day and all female mice fed up to 288 mg boron/kg/day survived treatment. Ninety percent of male mice survived exposure to 288 mg boron/kg/day, while 20% of males and 40% of females survived exposure to 577 mg boron/kg/day. Only at the high doses were the animals reported to be thin, haunchy and dehydrated with foot lesions and scaly tails. Hyperkeratosis and/or acanthosis of the stomach were reported in both sexes only at the highest dose, 577 mg boron/kg/day. Animals at all dose levels displayed nervousness and an increased incidence of extramedullary hematopoiesis of the spleen (NTP 1987). Reproductive effects observed in this study are discussed in Section D.3.a. of this report.

In a study by Weir and Fisher (1972) rats were fed diets containing borax or boric acid at 0, 52.5, 175, 525, 1750 or 5250 ppm as boron equivalent for 90 days. The physical appearance of rats receiving either compound at levels at and below 525 ppm boron were generally comparable to those of controls. Rats fed 1750 and 5250 ppm of boron were reported to display rapid respiration, inflamed eyes, swollen paws and desquamated skin on the paws and tails. All males at these two

doses had a shrunken scrotum during the last weeks of the study. Borax and boric acid up to 525 ppm boron did not affect growth, food consumption or food utilization efficiency of the rats. Boric acid reduced food consumption and growth in males and females at 1750 ppm boron; by contrast, exposure to borax at 1750 ppm boron reduced growth and food consumption in males only. Boron at up to 525 ppm did not produce any dose-related, statistically significant changes in organ weights. The study concluded that males fed either boron compound at 1750 ppm boron had statistically significant decreases in body weight and the weights of liver, spleen, kidneys and testes; borax at 1750 ppm boron also resulted in a reduction in brain weight, while boric acid lowered adrenal weights. In female rats fed 1750 ppm boron as borax or boric acid, weights of liver, spleen and ovaries were reduced, and boric acid reduced adrenal weights. Necropsies on rats that died (one each from 52.5 and 1750 ppm boron levels of borax and all rats at 5250 ppm boron level of borax and boric acid) showed congestion of liver and kidneys and bright red lungs. In several rats, brains were swollen, gonads were small and the pancreas was thickened. Reproductive effects reported in this study are discussed in Section D.3.a of this report.

Weir and Fisher (1972) also fed beagle dogs borax or boric acid in the diet at 0, 17.5, 175, or 1750 ppm as boron equivalent for 90 days. All dogs, with one exception, fed either boron compound at each dose level were normal in appearance, behavior, elimination, body weight and food consumption. The study noted that a single male dog in the high dose group which died at day 68 of the study showed congested kidneys and severe congestion of mucosa of the intestines. Neither borax nor boric acid at 17.5 or 175 ppm as boron equivalent produced any statistically significant, dose-related changes in organ weights or organ-to-body weight ratios. The study reported that borax and boric acid at only the high dose caused statistically significant decreases in thyroid and testes-to-body weight ratios in male dogs. At 1750 ppm boron as borax, increases in brain-to-body weight ratios reportedly occurred, and boric acid at the same level reportedly resulted in increases in liver-to-body weight ratios in both sexes. Reproductive effects observed in this study are also discussed in Section D.3.a of this report.

Rats receiving a diet containing borax or boric acid at 117 or 350 ppm as boron equivalent for 2 years were generally comparable in appearance and behavior with that of the controls, and no histologic changes were seen in the organs of these rats. In rats fed the very high level of 1170 ppm boron for two years, coarse hair coats, scaly tails, swelling and desquamation of the paws, shrunken appearance of the scrotum of males, inflamed eyelids and bloody discharge of the eyes were

observed (Weir and Fisher 1972). Reproductive effects reported in this study are also discussed in Section D.3.b of this report.

In male mice fed 48 and 96 mg boron/kg/day (as boric acid) for 103 weeks, mortality was 40% and 56%, respectively, compared to 18% in the control group (NTP 1987; ATSDR 1990). Mortality in female mice was 30% and 24% at the low and high dose levels, respectively, compared to 34% in the untreated control. A dose-related decrease in body weight gain was apparently observed in both male and female mice after 30 weeks of dosing. Administration of boric acid to high-dose male mice caused interstitial cell hyperplasia and testicular atrophy; these effects are discussed in Section D.3.b of this report.

Rats exposed to 77 mg/m^3 boron oxide aerosol for as long as 24 weeks were unaffected. Rats exposed to 470 mg/m^3 boron oxide aerosol for 10 weeks reportedly developed reddish exudate from the nose, but no deaths or other signs of intoxication were observed (Wilding et al. 1959). Dogs exposed to 57 mg/m^3 for 23 weeks exhibited increased urine volume, urine acidity and creatinine clearance.

Shuler and Nielsen (1988) have reported that boron and the interaction between boron and magnesium may affect major mineral metabolism in chicks and rats.

b) Human Data

Some data on human exposure to boron suggest a positive effect from boron supplementation in the diet. Post-menopausal women were fed a diet low in boron for 119 days, after which they were administered a boron supplement of 3 mg/day (Nielsen et al. 1987). The boron supplement markedly affected several indices of mineral metabolism, including urinary excretion of calcium and magnesium. Elevation in serum steroids (17B-estradiol and testosterone) was also observed. These findings suggest that supplementation of a low-boron diet with an amount of boron commonly found in diets high in fruits and vegetables may help prevent calcium loss and bone demineralization in post-menopausal women.

Infants who ingested 4 to 30 grams boric acid for 4 to 10 weeks or 9 to 125 grams borax for 5 to 12 weeks developed seizure disorders (O'Sullivan and Taylor 1983; Gordon et al. 1973). Workers employed in the borax industry for 11 years with mean exposures of 4.1 mg/m^3 to boron oxide and boric acid dusts experienced dryness of the mouth, nose, or throat, sore throat, productive cough, and eye irritation (Garabrant et al. 1984; Garabrant et al. 1985).

3. Reproductive and Developmental Toxicity

a) Subchronic Toxicity

Treinen and Chapin (1991) examined the development of testicular lesions in rats fed the extremely high level of 9000 ppm boric acid for up to 28 days. After 7 days, inhibition of spermiation was reported in 10-30% of seminiferous tubules. As exposure continued, exfoliation of germ cells into the tubular lumen was noted. After 28 days of dosing, extreme epithelial disorganization and germ cell loss were seen. A decrease in basal testosterone levels and an absence of selective accumulation of boron in the testis was also reported.

Weir and Fisher (1972) did not report any reproductive effects in rats fed diets containing borax or boric acid at 52.5 or 175 ppm as boron equivalent for 90 days. Partial testicular atrophy was reported in rats fed 525 ppm boron as borax or boric acid and severe testicular atrophy was reported in dogs fed 1750 ppm boron as borax or boric acid (Weir and Fisher 1972). No reproductive effects were evident in rats administered up to 6 mg boron/liter of borax (0.426 mg boron/kg/day) in drinking water for 90 days (Dixon et al. 1976). One study claimed that seminal vesicles of rats provided with drinking water containing 150 or 300 mg boron/liter as borax for 70 days were atrophic (Seal and Weeth 1980). Impaired spermatogenesis was reported in rats receiving the higher dose (Seal and Weeth 1980). Reproductive effects were not observed in male mice fed up to 72 mg boron/kg/day as boric acid for 13 weeks, but degeneration or atrophy of the seminiferous tubules was noted in mice fed the extremely high concentration of 144 mg boron/kg/day (5000 ppm boric acid) for 13 weeks (NTP 1987). Male rats exposed to 500 ppm boron as borax in their diet for periods of 30 and 60 days did not demonstrate any significant adverse effects other than a decrease in epididymal weights. Rats exposed to 1000 and 2000 ppm boron displayed a decrease in testicular weight and a statistically significant loss of germinal elements after 60 days of treatment (Dixon et al. 1979). Lee et al. (1978) reported that testes of rats fed 500 ppm boron as borax showed no morphological changes at 30 or 60 days. Marked reduction of spermatocytes, spermatids and mature spermatozoa was reported after 30 days exposure to 1000 ppm boron as borax. By 60 days, most germinal elements were absent. In testes from rats treated with 2000 ppm boron, complete germinal aplasia was seen at 60 days. Serial matings in rats treated at the 1000 ppm dose level revealed a reduced pregnancy rate for 3 weeks following 30 days on the boron diet and for 4 weeks following 60 days of treatment (Lee et al. 1978). At 2000 ppm boron, pregnancy rates were significantly reduced after 8 weeks with only a partial recovery observed at week 9-10, following 30 days of

boron treatment. No pregnancies were observed during the 12-week mating period after 60 days of boron treatment at 2000 ppm.

b) Chronic Toxicity

In a study by Weir and Fisher (1972), boron as boric acid or borax was added to the diets of dogs at 0, 58, 117, or 350 ppm for 2 years, or 1170 ppm for 38 weeks. Neither borax nor boric acid at the 58, 117 or 350 ppm boron dose levels produced any testicular changes. At the highest dose (1170 ppm), severe testicular atrophy and spermatogenic arrest occurred after 26 weeks of treatment. Rats receiving a diet containing borax or boric acid at 117 or 350 ppm as boron equivalent showed no histologic changes in the organs. At the highest dose, 1170 ppm, atrophic testes, decreased testes weights and testes-to-body weight ratios were observed. In reproduction studies there were no adverse effects on the reproduction of rats receiving a diet containing either borax or boric acid at 117 or 350 ppm as boron equivalent. Borax and boric acid at 1170 ppm boron for 14 weeks led to sterility, with lack of viable sperm in atrophied testes of all males and decreased ovulation in the majority of females at this dose level (Weir and Fisher 1972).

Male mice fed 48 mg boron/kg/day for 103 weeks experienced no increased incidence of testicular atrophy or interstitial cell hyperplasia. However, in mice fed 96 mg boron/kg/day, testicular atrophy and interstitial cell hyperplasia were observed. The testicular atrophy was characterized by variable loss of spermatogonia, primary and secondary spermatocytes, spermatids and spermatozoa from the seminiferous tubules (NTP 1987).

Krasovskii et al. (1976) reported a tendency toward reduction in sexual function in men using water with high boron content (0.3 mg/kg). Men working in boric acid production facilities reportedly showed weakened sexual activity, decreased seminal volume, low sperm count and motility, and increased seminal fructose (Taresenko et al. 1971).

4. Mutagenicity

No evidence of the mutagenicity of boron was found in the available literature.

5. Carcinogenicity

In mice fed up to 96 mg boron/kg/day as boric acid for 103 weeks, the number of tumors observed did not differ significantly from the control (NTP 1987). No increased tumor incidence was observed in mice provided with water containing 5 ppm boron throughout their lifespan (Schroeder and Mitchener 1975). Two-year studies in rats fed

up to 58.5 mg boron/kg and dogs fed up to 8.8 mg/kg/day (as borax or boric acid) also revealed no increase in tumor incidences (Weir and Fisher 1972). No epidemiological studies were located associating cancer and boron exposure.

E. Pharmacokinetics and Metabolism

Two studies indicate that boron is readily absorbed in humans by the gastrointestinal tract (Kent and McCane 1941; Aas Jansen et al. 1984). In a study by Kent and McCane, two women ingested 325 mg boron over a 3-day period. Approximately 93% of the boron was recovered in the urine in the first week. Aas Jansen et al. (1984) administered boric acid to human males via drinking water or ingestion of food. Approximately 93% of the boron administered was excreted in urine within 96 hours following boron intake. Urinary excretion studies in humans suggest there is very little absorption of boron through intact skin, although studies in rabbits suggest that boron is readily absorbed following contact with damaged skin (Draize and Kelley 1959). Pfeiffer et al. (1945) established through direct titration of boric acid in the urine that "practically all" of the boron present in the urine of dogs was in the form of boric acid, suggesting that the compound is probably not converted to other metabolites. Boron is excreted primarily in urine. Tipton et al. (1966) demonstrated that approximately 98% of 350-420 micrograms (μg) boron ingested daily in foodstuffs was excreted through the kidneys, while less than 2% was excreted in the feces.

F. Development of RfD

There is some uncertainty associated with the development of an Oral Reference Dose (RfD) applicable to the human population based on data from experiments in laboratory animals. Because of limitations inherent in studying the toxic effects of chemicals in humans, much of our knowledge of the toxicity of chemicals comes from experiments in laboratory animals. Experimental animal data have been relied on for many years by regulatory agencies and other expert groups for assessing the hazards or safety of human exposure to chemicals. This reliance has been supported in general by the overall similarities in anatomy and physiology between humans and other mammals. However, there are differences in response and the exposure conditions (dose, level and duration), between humans and the species for which experimental toxicity data are generally available. Because of this, regulatory agencies and others generally use uncertainty factors or safety factors in determining safe exposure levels of non-carcinogens, to guard against the possibility that humans are more sensitive than the most sensitive experimental animal species tested. Such uncertainty factors have been used to derive the RfD for boron.

The USEPA (1990) has recommended an RfD (for both subchronic and chronic exposure) of 0.09 mg/kg/day based on a no-observed-adverse-effect level (NOAEL) from the Weir and Fisher (1972) study. In this study, boron as boric acid or borax was added to

the diets of dogs at 0, 58, 117 or 350 ppm for 2 years, or 1170 ppm for 38 weeks. Only at the highest dose, severe testicular atrophy and spermatogenic arrest occurred after 26 weeks of treatment. These changes were not observed at the 350 ppm (8.8 mg/kg/day) dose level. Using the NOAEL of 8.8 mg/kg/day and applying an uncertainty factor of 100 (10 for interspecies extrapolation and 10 to protect sensitive individuals), USEPA derived an RfD of 0.09 mg/kg/day.

G. Acceptable Drinking Water Level

A variety of studies have reported ranges of daily intakes of boron. Ploquin (1967) reported a daily intake of boron near 7 mg, excluding beverages. Tipton et al. (1966) reported mean boron intakes of 0.42 and 0.35 mg/day for two adults consuming self-selected diets, including beverages. Average daily intakes of 1.7 mg boron from food and beverages were reported by Varo and Koivistoinen (1980) for Finnish people. Zook and Lehmann (1965) obtained an overall average boron content of 3.1 mg/day in total diets, including food and drinking water, with a range of 2.1 to 4.3 mg/day. Hamilton and Minski (1972-1973) reported a mean of 2.8 $\pm$ 1.5 mg boron/day for English total diets. The World Health Organization (WHO) has estimated the average daily intake of boron as 3.0 mg (WHO 1973). Nielsen (1988b) reported that "recent surveys indicate dietary intakes of 1.7 to 7 mg boron/day are average." This range seems to be representative of the studies described above.

By using the midpoint of the range reported by Nielsen (1988b), 4.4 mg boron/day, and an adult body weight of 70 kg, a daily dietary dose for boron of 0.06 mg/kg/day is calculated by ENVIRON. As discussed in the preceding section, the USEPA has established an oral RfD for boron of 0.09 mg/kg/day. Therefore, the RfD adjusted for dietary intake of boron (oral RfD minus dietary dose) is 0.03 mg/kg/day. Using the assumption for body weight described above, and a value for water consumption by an adult of 2 liters/day, the adjusted RfD corresponds to an acceptable level of 1.05 mg/L in drinking water (see Equation 1).

(1) $$\frac{(x\ mg/L)(2\ L/day)}{70\ kg} = 0.03\ mg/kg/day$$

$$x = 1.05\ mg/L$$

In addition, the USEPA Office of Water has published a draft Drinking Water Health Advisory for boron, which includes a Lifetime Health Advisory (HA) (USEPA 1991). The Lifetime HA represents that portion of an individual's total exposure that is attributed to

drinking water and is considered protective of noncarcinogenic adverse health effects over a lifetime exposure. To derive the Lifetime HA, the USEPA RfD for boron, 0.09 mg/kg/day was converted to a Drinking Water Equivalent Level (DWEL) by multiplying the RfD by the body weight of an adult and dividing by the assumed daily water consumption of an adult. This DWEL represents the media-specific lifetime exposure level, assuming 100% exposure from that medium, at which adverse noncarcinogenic health effects would not be expected to occur. The Lifetime HA in drinking water alone is determined by factoring in other sources of exposure, the relative source contribution (RSC). The RSC from drinking water can be based on actual dietary intake data, as described in the preceding paragraph, or if data are not available, an RSC of 20% is assumed. Equation 2 describes the process by which the Lifetime HA for boron was calculated by the USEPA.

(2) $$DWEL = \frac{(0.09\ mg\ B/kg/day)(70\ kg)}{2\ L/day} = 3.15\ mg\ B/L$$

$$Lifetime\ H.A. = (3.15\ mg\ B/L)(20\%) = 0.63\ mg\ B/L$$

Rounding of Health Advisory levels is traditionally done by the USEPA, and according to Robert Cantilli, a biologist with USEPA's Office of Water, the Lifetime HA of 0.63 mg B/L was rounded to a value of 1 mg B/L (Cantilli 1991). This value agrees with the value of 1.05 mg/L calculated by ENVIRON, the permissible criteria reported by the U.S. Department of the Interior (1968) for raw surface water (1 mg/L), and criteria proposed by Eisler of the U.S. Fish and Wildlife Service (1990) for the protection of human health (1.0 mg/L).

H. Environmental Toxicity

1. Wildlife and Domestic Animals

Smith and Anders (1989) fed adult mallard ducks (*Anas platyrhynchos*) diets supplemented with 0, 30, 300 or 1000 ppm boron as boric acid. After receiving the experimental diets for 3 weeks, males and females were paired. The resulting ducklings continued on the same diet for 21 days. The hatching success of eggs laid by mallards in the two lower dose groups did not differ from that of the control group. Boron did not produce any overt signs of toxicosis in either adult or young mallards. The hatching success of fertile eggs laid by mallards which had received 1000 ppm boron was reduced

to approximately 52% of that of controls. Hatching weights, duckling survival and duckling weight gain were also reportedly reduced by 1000 ppm boron. Boron concentrations in mallard egg, liver and brain tissues were dose-related.

In a similar study (Hoffman et al. 1990), day-old mallard ducklings received diets containing 0, 100, 400, or 1600 ppm boron as boric acid. None of the dietary levels of boron had a significant effect on survival. The females in all three treatment groups exhibited delayed growth and lower growth rates compared to controls. In both sexes, the highest concentration of boron caused 10% mortality, decreased overall growth and the rate of growth. The groups exposed to doses of 400 and 1600 ppm boron reportedly exhibited statistically significant effects on brain biochemistry, including a decrease in brain adenosine triphosphate (ATP) concentration. Ducklings exposed to boron at these concentrations also reportedly exhibited altered behavior such as an increased amount of time spent resting and reduced amount of time spent bathing and standing compared to controls.

Birge and Black (1977) injected chicken eggs with doses of boric acid and borax at concentrations of 0.01 to 50 ppm boron. Hatching frequencies of control eggs ranged from 78 - 89%. The hatchability of eggs treated with boric acid at exposure levels of 0.01, 1.0 and 50 ppm was 92%, 44% and 0%, respectively. The frequency of chicks with embryonic anomalies (teratogenic chicks) surviving treatment with boric acid ranged from 4% at 0.50 ppm boron to 10% at 10 ppm. Treatment with borax at 0.01, 1.0 and 50 ppm boron gave hatching frequencies of 95%, 49%, and 0%, respectively. Frequencies of teratogenesis following treatment with borax ranged from 42% at 25 ppm to 2-3% at 0.10 to 0.25 ppm. Combining teratogenesis and lethality, the LC_{50} values for boric acid and borax were approximately 1.0 ppm and 0.5 ppm boron, respectively. The LC_1 values appeared to be close to 0.01 ppm boron. No teratogenic development was observed at this exposure level and hatchability was 92-95% of that observed for controls (Birge and Black 1977). Hens fed 0.5% boric acid (875 ppm) in their food ceased egg production after 6 days, but returned to normal production in 14 days when the boron was omitted (Hove et al. 1939).

A maximum acceptable concentration of 5.0 mg boron/L in livestock drinking water has been proposed by the USEPA (1973), and also by the U.S. Fish and Wildlife Service (1990) based on the following studies. Boron has been added to the diets of lactating dairy cattle at 145 to 157 ppm in the form of borax with no adverse effects (Owen 1944). Green and Weeth (1977) gave Hereford heifers tap water (0.8 ppm boron) supplemented with 150 or 300 ppm boron added as borax. The heifers were fed hay that contained 38.3 ppm boron. Exposure periods lasted 30 days. Heifers exposed to boron at both concentrations reportedly exhibited reduced feed consumption, weight loss, swelling and irritation in the legs and around dew claws, and a decrease in

hematocrit and hemoglobin. At 300 ppm, lethargy and diarrhea were observed. The authors concluded that 300 ppm boron is not acutely toxic to heifers when consumed via drinking water, and that the safe tolerance concentration probably lies between 40 and 150 ppm. Weeth et al. (1981) offered yearling beef heifers drinking water to which 0, 15, 30, 60, or 120 mg boron/L as borax was added for 10 days. No effects on feed or water consumption or any overt signs of toxicosis were seen. Sheep have been found to develop enteritis when exposed to naturally occurring boron-enriched soils (30-300 mg boron/kg) and associated water levels of 1-20 mg boron/L (Koval'skii et al. 1965).

2. Plants

Boron is an essential nutrient for the growth of higher plants, but boron in excess of threshold concentrations may be phytotoxic. Plants vary greatly in their sensitivity to boron toxicity; the range of concentrations within which boron is essential to some plants overlaps with that within which it is toxic to others (U.S. Fish and Wildlife Service 1990; Butterwick et al. 1989). The ratio of toxic to sufficient levels of boron is smaller than that for any other nutrient element (Reisenauer et al. 1973). The amount of boron taken up by plants differs between species and depends on the stage of growth. A variety of soil and environmental factors also affect the uptake of boron by plants. Boron retention in soil depends on boron concentration in the soil solution, soil pH, texture, organic matter, cation exchange capacity, type of clay, moisture content, wetting and drying cycles, and temperature. Soil pH is one of the most important factors affecting boron uptake by plants. In general, boron becomes less available to plants with increasing soil pH (Gupta 1979).

Boron at less than 0.3 mg/L is recommended by the U.S. Fish and Wildlife Service for the protection of sensitive crops (U.S. Fish and Wildlife Service 1990). The soil boron content necessary for optimum growth of a variety of crops is presented in Table 2. The threshold concentration range, the maximum concentration that a plant species tolerates without manifesting visible injury symptoms and/or a decrease in yield, is shown in Table 3 for a variety of crop species. Species especially sensitive to the effects of boron in soil water include citrus fruits, stone fruits, and nut trees; semitolerant species include most vegetables; tolerant species include cotton and tomatoes. The toxic symptoms of boron include stunted growth, malformations of leaves, browning and yellowing, chlorosis, necrosis, increased sensitivity to mildew, wilting, and inhibition of pollen germination and pollen tube growth (USEPA 1975; U.S. Fish and Wildlife Service 1990). Critical concentrations of boron in irrigation water were developed by Eaton (1935) on the basis of relative sensitivity of various species to boron; these values are shown in Table 4. Further irrigation water quality guidelines are also presented in Table 4; some of these guidelines consider the type of

soil and the duration of the irrigation practice, which affects the accumulation of boron in the soil (Butterwick et al. 1989).

3. **Fresh Water Aquatic Organisms**

As discussed previously, the predominant species of boron in most fresh water systems (pH 6-9) is undissociated boric acid, regardless of its initial formulation (Butterwick et al. 1989, Hem 1970). Therefore, the formulation of boron should not significantly influence its aquatic effects at naturally occurring pH levels (Procter & Gamble 1987). Table 5 summarizes the toxicological effects of boron on aquatic life other than fish. Bringmann (1978) reported the effects threshold for the protozoan *Entosiphon sulcatumm* as 1.0 mg boron/L; at this concentration, cell replication was reduced by 5%. McKee and Wolf (1963) reported the cladoceran *Daphnia magna* to be relatively sensitive to the effects of boron. Lewis and Valentine (1981) performed 48-hour static and 21-day static renewal tests with boric acid to determine its acute and chronic effects to *D. magna*. The 48-hour LC_{50} value was 226 mg boron/L and in the 21-day chronic test, the LC_{50} was 53.2 mg boron/L. Based upon the most sensitive parameters, mean brood size and total young produced, the no-observed-effect concentration (NOEC) was 6 mg boron/L, and the no-effect concentration was between 6 and 13 mg boron/L. A similar investigation of *Daphnia* conducted in water from Lake Huron resulted in similar effects values (Gersich 1984). The 21-day LC_{50} was reported as 52.2 mg boron/L, and the NOEC and LOEC were reported as 6.4 and 13.6 mg boron/L, respectively.

In an investigation of the effects of boron on early life stages of amphibians, developmental stages of the leopard frog (*Rana pipiens*) and embryos and larvae of Fowler's toad (*Bufo fowleri*) received continuous exposure to boron as boric acid or borax at two levels of water hardness for 7.5 days (Birge and Black 1977). Posthatch LC_{50} values for the leopard frog were 130 and 135 ppm for boric acid in soft and hard water, respectively, and 47 and 54 ppm for borax in soft and hard water, respectively. Posthatch LC_{50} values for Fowler's toad were 145 and 123 ppm for boric acid in soft and hard water, respectively.

Early life stages of nonsalmonid fish species are relatively resistant to aqueous exposure to boron. Results of an unpublished study (Procter and Gamble 1979) of boric acid administered to fathead minnow egg-fry indicated a 30-day LOEC (reduction in growth) of 24 mg boron/L and a 60-day LOEC (reduction in fry survival) of 88 mg boron/L. The LOECs for embryo-larval stages of the channel catfish and goldfish administered boric acid or borax ranged from 1.04 to 25.9 mg boron/L and 8.33 to 48.75 mg boron/L, respectively (Birge and Black 1977). A LOEC of approximately 12 mg boron/L was reported for largemouth bass administered boric acid (Birge and

TABLE 2 Boron Content of Soils for Optimum Growth[a]		
0.1 mg B/kg	0.1 to 0.5 mg B/kg	0.5 mg B/kg
Small grain	Tobacco	Apple
Corn	Tomato	Alfalfa
Soya bean	Lettuce	Clovers
Pea and bean	Peach	Beets
Strawberry	Pear	Turnips
Potato	Cherry	Cruciferae
Grass	Olive	Asparagus
Flax	Pecan	Radish
	Cotton	Celery
	Sweet potato	Rutabaga
	Peanut	
	Carrot	
	Onion	

[a] Source: Butterwick et al. 1989

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TABLE 3
Threshold Concentration Range for Boron in Soil Water, Field Capacity Basis, According to Crop Species[a]

Crop Species	Threshold concentration range (mg B/L)
Sensitive crops	**(0.30-1.01)**
Lemon (*Citrus limon*)	0.30-0.50
Blackberry (*Rubus* sp.)	↓
Avocado (*Persea americana*)	0.50-0.80
Orange (*Citrus sinensis*)	↓
Grapefruit (*Citrus paradise*)	
Apricot (*Prunus armeniaca*)	
Peach (*Prunus persica*)	
Cherry (*Prunus avium*)	
Plum (*Prunus domestica*)	
Persimmon (*Diosypysos kaki*)	
Fig (*Ficus carica*)	
Grape (*Vitis vinifera*)	
Walnut (*Juglans regia*)	
Pecan (*Carya illinoensis*)	
Cowpea (*Vigna sinensis*)	
Onion (*Allium cepa*)	
Garlic (*Allium sativum*)	0.80-1.01
Sweet potato (*Ipomoea batatas*)	↓
Wheat (*Triticum aestivum*)	
Mung bean (*Phaseolus aureux*)	
Strawberry (*Fragaria* sp.)	
Kidney bean (*Phaseolus vulgaris*)	
Lima bean (*Phaseolus lunatus*)	↓
Semitolerant crops	**(1.01-4.00)**
Sesame (*Sesamum indicum*)	1.01-2.05
Red pepper (*Capsicum annum*)	↓
Pea (*Pisum sativum*)	
Carrot (*Daucus carota*)	
Radish (*Raphanus sativus*)	
Potato (*Solanum tuberosum*)	
Cucumber (*Cucumis sativus*)	
Lettuce (*Lactuca sativa*)	
Cabbage (*Brassica oleracea*)	
Celery (*Cepium graveolens*)	
Turnip (*Brassica rapa*)	
Barley (*Hordeun vulgare*)	↓

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TABLE 3
Threshold Concentration Range for Boron in Soil Water, Field Capacity Basis, According to Crop Species[a]

Crop Species	Threshold concentration range (mg B/L)
Corn (*Zea mays*)	2.05-4.00
Artichoke (*Cynara scolymus*)	↓
Tobacco (*Nicotiana tabaacum*)	↓
Sweet clover (*Mehlotus indica*)	↓
Squash (*Cucurbita pepo*)	↓
Muskmelon (*Cucumis melo*)	↓
Tolerant crops	**(4.00-15.03)**
Sorghum (*Sorghum bicolor*)	4.00-6.05
Alfalfa (*Medicago sativa*)	↓
Purple betch (*Vicia benghalensis*)	↓
Oat (*Avena vulgare*)	↓
Parsley (*Petroselium crispum*)	↓
Red beet (*Beta vulgaris*)	↓
Tomato (*Lycopersicon lycopersicum*)	↓
Sugarbeet (*Beta vulgaris*)	↓
Cotton (*Gossypium hirsutum*)	6.05-10.05
Asparagus (*Asparagus officinalis*)	10.05-15.03

[a] Source: Keren and Bingham (1985), as referenced in Butterwick et al. (1989)

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TABLE 4
Irrigation Water Quality Guidelines for Boron[a]

Source	Criterion	Irrigation water boron limit (mg B/liter)
	Nature of crop	
Eaton (1935)	Sensitive	0.3-1
	Semitolerant	1-2
	Tolerant	2-4
FAO (UNESCO) (1976)	Sensitive	0.5-1
	Semitolerant	1-2
	Tolerant	2-10
Israel	All crops	0.7
	Nature of soil/time	
NAS and NAE (1972)	All soils/long-term Fine textured soils for 20 years	1.0 2.0
EPA (1973)	All soils/long-term Fine textured neutral and alkaline soils for 20 years	0.75 2.0
	Degree of Problem	
Ayers and Westcot (1976)	No problem	<0.5
	Increasing problem	0.5-2.0
	Severe problem	2.0-10.0

[a] Source: Gupta (1983), as referenced in Butterwick et al. (1989)

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TABLE 5
Summary of Boron Toxicity Data for Aquatic Life Other Than Fish

Test organism	Boron compound tested	Type of test	Water quality characteristics	Test response(s) reported	Boron concentration (mg B/liter)	Reference
Invertebrates						
Daphnia magna Straus	Boric acid H_3BO_3	Static	Lake Huron water; 20°C; pH 6.7-8.1; hardness 150 mg/liter	48-hr LC_{50}	133	Gersich (1984)
D. magna Straus	Boric acid H_3BO_3	Static renewal (3 times weekly)	Lake Huron water; 20°C; pH 7.3-8.0; hardness 150 mg/liter	21-day LC_{50} 21-day NOEC-LOEC	52.2 6.4-13.6	Gersich (1984)
D. magna Straus	Boric acid H_3BO_3	Static	Carbon-filtered well water; 19.2°C; pH 7.1-8.7; hardness 166 mg/liter	48-hr LC_{50}	226	Lewis and Valentine (1981)
D. magna Straus	Boric acid H_3BO_3	Static (3 times weekly)	Carbon-filtered well water; 19.2°C; pH 7.1-8.7; hardness 166 mg/liter	21-day LC_{50} 21-day No-effect concentration	53.2 6-13	Lewis and Valentine (1981)
D. magna	Sodium perborate $Na_2BO_3 \cdot 4\ H_2O$	--	Lake Erie H_2O; 25°C	Threshold concentration for immobilization	<0.38 estimated to be 0.19; (<5.2; estimated to be 2.6)	McKee and Wolf (1963)

TABLE 5
Summary of Boron Toxicity Data for Aquatic Life Other Than Fish

Test organism	Boron compound tested	Type of test	Water quality characteristics	Test response(s) reported	Boron concentration (mg B/liter)	Reference
D. magna	Sodium tetraborate $Na_2B_4O_7 \cdot 10\ H_2O$	--		Threshold concentration for immobilization	<27.2; estimated to be 13.6; (<240; estimated to be 120)	McKee and Wolf (1963)
Protozoan (*Entosiphon sulcatum*)	Sodium tetraborate $Na_2B_4O_7 \cdot 10\ H_2O$	Static	Culture medium adjusted to pH 6.9; 25°C	Toxicity threshold (measured as a 5% reduction in cell replication after 72 hr)	1.0	Bringmann (1978)
Mosquito larvae (*Anopheles quadrimaculatus*)	Boric acid $H_3BO_3 \cdot H_2O$	--	--	100% mortality after 25 hr	125	Fay (1959)
				92% mortality after 48 hr	25	
Amphibians						
Toad (*Bufo vulgaris formosus*)	Boric acid H_3BO_3	Static; embryos exposed to B for 24 hr at various embryonic stages and then cultured in tap water until 14 days past fertilization	Boric acid solutions prepared in tap water	Teratogenic defects and reduced survival	1747 (1% solution)	Takeuchi (1958)

TABLE 5
Summary of Boron Toxicity Data for Aquatic Life Other Than Fish

Test organism	Boron compound tested	Type of test	Water quality characteristics	Test response(s) reported	Boron concentration (mg B/liter)	Reference
Fowler's toad (*Bufo fowleri*), embryo-larval stages	Boric acid H_3BO_3	Flowthrough	Reconstituted water, hardness 50 mg $CaCO_3$/liter	7-day LC_1; LC_{50}	25; 145	Birge and Black (1977)
Fowler's toad (*Bufo fowleri*), embryo-larval stages	Boric acid H_3BO_3	Flowthrough	Reconstituted water, hardness 200 mg $CaCO_3$/liter	7-day LC_1; LC_{50}	5; 123	Birge and Black (1977)
Amphibians						
Leopard frog (*Rana pipiens*), embryo-larval stages	Boric acid H_3BO_3	Flowthrough	Reconstituted water, hardness 50 mg $CaCO_3$/liter	7-day LC_1; LC_{50}	13; 130	Birge and Black (1977)
Leopard frog (*Rana pipiens*), embryo-larval stages	Boric acid H_3BO_3	Flowthrough	Reconstituted water, hardness 200 mg $CaCO_3$/liter	7-day LC_1; LC_{50}	22; 135	Birge and Black (1977)
Leopard frog (*Rana pipiens*), embryo-larval stages	Borax $Na_2B_4O_7 \cdot 10\ H_2O$	Flowthrough	Reconstituted water, hardness 50 mg $CaCO_3$/liter	7-day LC_1; LC_{50}	5; 47	Birge and Black (1977)
Leopard frog (*Rana pipiens*), embryo-larval stages	Borax $Na_2B_4O_7 \cdot 10\ H_2O$	Flowthrough	Reconstituted water, hardness 200 mg $CaCO_3$/liter	7-day LC_1; LC_{50}	3; 54	Birge and Black (1977)

TABLE 5
Summary of Boron Toxicity Data for Aquatic Life Other Than Fish

Test organism	Boron compound tested	Type of test	Water quality characteristics	Test response(s) reported	Boron concentration (mg B/liter)	Reference
Plants						
Spiked or Eurasian watermillfoil (*Myriophyllum spicatum*)	Tetraborate salt B_4O^{2-}	--	Fresh water	50% inhibition of roots weight after 32 days	40.3	Stanley (1974)
Chlorella algae (*C. vulgaris*)	--	--	--	Toxic	50	Bowen and Gauch (1966)
Chlorella algae (*C. protothicoides* and *C. emersanii*)	--		--	Toxic	100	Bowen and Gauch (1966)

Source: Butterwick et al. 1989.

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Black 1981). Birge and Black (1977) have reported that embryonic mortality and teratogenesis of aquatic vertebrates is greater in hard water than in soft water, but larval mortality of fish and amphibians is usually higher in soft water.

The early life stages of rainbow trout appear to be the most sensitive to boron. Even based on the data presented below for the most sensitive species, rainbow trout, a boron criterion of 1.0 mg/L is recommended by the U.S. Fish and Wildlife Service for the protection of aquatic life (U.S. Fish and Wildlife Service 1990). In a study by Birge and Black (1977), developmental stages of rainbow trout were exposed for 28 days to boron concentrations (boric acid and borax) ranging from 1 part per billion (ppb) to 200 ppm. A LOEC of 0.1 mg boron/L was reported in reconstituted water. Boron in natural waters is less toxic to embryo-larval stages of rainbow trout (Procter & Gamble 1987). Under natural water exposures an LOEC of 1.0 mg boron/L was recorded by Birge et al. (1984) and Procter and Gamble (1987), and 0.75 mg boron/L (natural background boron) did not affect early life stages of rainbow trout. Another early life stage rainbow trout study by Procter and Gamble conducted in natural water indicated no impairment of early life stages at 17.0 mg boron/L. Results of all the trout toxicity tests indicate that it takes greater than 1.0 mg boron/L to get a 10% increase in the control adjusted mortality (Butterwick et al. 1989). Procter and Gamble concluded that the low-level effects observed in reconstituted laboratory water are not predictive of the much higher levels at which effects were first observed under natural exposure conditions. Further support for this theory is available in documented reproducing populations of native wild and hatchery-raised rainbow trout which have been observed to exist in surface water containing up to 13 mg boron/L (Bingham 1982.) Table 6 presents a summary of boron toxicity data for fresh water fish.

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TABLE 6
Summary of Boron Toxicity Data for Fresh Water Fish

Test organism	Boron compared tested	Type of test	Water quality characteristics	Test response(s) reported	Boron concentration (mg B/liter)	Reference
Bluegill sunfish (*Lepomis macrochirus*), average size 7 cm, 5 g	Sodium tetraborate $Na_2B_4O_7 \cdot 10\ H_2O$	Static	Dechlorinated Philadelphia tap water; 20°C; pH 6.9-7.5; alkalinity 33-81 mg/liter; hardness 84-163 mg/liter	24-hr TLm[1]	4.6	Turnbull et al. (1954)
Bluegill sunfish (*L. macrochirus*), average size 7 cm, 5 g	Boron trifluoride BF_3	Static	Dechlorinated Philadelphia tap water; 20°C; alkalinity 1750 mg/liter	24-hr TLm	2389	Turnbull et al. (1954)
Coho salmon (*Oncorhynchus kisutch*), alevins, 0.19-0.7 g	Sodium metaborate $Na_2B_4O_7 \cdot 8\ H_2O$	Static renewal (daily)	Well water; 11.0°C; hardness 47 mg/liter	283-hr LC_{50}	113	Thompson et al. (1976)
Fathead minnow (*Pimephales promelas*), eggs and fry	Boric acid H_3BO_3	Flowthrough early life stage	Well water; 25°C; pH 7.1-7.9; alkalinity 33-38 mg/liter, hardness 38-46 mg/liter	30-day NOEC-LOEC (reduction in growth) 60-day NOEC-LOEC (reduction in fry survival)	14-24 24-88	Procter & Gamble (1979) (unpublished)
Minnow	Boric acid H_3BO_3	--	20°C; distilled H_2O 20°C; hard H_2O	6-hr minimum lethal dose 6-hr minimum lethal dose	3.145-3.319 3.319-3.407	NAS (1973); McKee and Wolf (1963)
Minnow	Sodium tetraborate $Na_2B_4O_7 \cdot 10\ H_2O$	--	19°C; distilled H_2O 17°C; hard H_2O	Minimum lethal dose Minimum lethal dose	340-374 793-850	McKee and Wolf (1963)

TABLE 6
Summary of Boron Toxicity Data for Fresh Water Fish

Test organism	Boron compared tested	Type of test	Water quality characteristics	Test response(s) reported	Boron concentration (mg B/liter)	Reference
Mosquito fish (*Gambusia affinis*), adult females	Boric acid H_3BO_3	Static	20-23°C; pH 5.4-7.3; turbidity 210-250 mg/liter	24-hr TLm 48-hr TLm 96-hr TLm No mortalities in 96-hr	3145 1834 978 <314	Wallen et al. (1957)
Mosquito fish (*G. affinis*), adult females	Sodium tetraborate $Na_2B_4O_7 \cdot 10\ H_2O$	Static	22-26°C; pH 8.6-9.1; turbidity 410-650 mg/liter	24-hr TLm 48-hr TLm 96-hr TLm 144-hr TLm No mortalities	1360 929 408 215 <204	Wallen et al. (1957)
Rainbow trout (*Salmo gairdneri*)	Boric acid H_3BO_3	--	--	Darkening of skin, immobilization, and loss of equilibrium	874 13,976	Wurtz (1945)
Trout, fingering	Sodium perborate $NaBO_3 \cdot 4\ H_2O$	--	--	80% mortality	23.7	McKee and Wolf (1963)
Channel catfish (*Ictalurus punctatus*), embryo-larval stages	Boric acid H_3BO_3	Flowthrough	Reconstituted water; hardness 50 mg $CaCO_3$/liter	9-day NOEC-LOEC	1.01-5.42	Birge and Black (1977)
Channel catfish (*Ictalurus punctatus*), embryo-larval stages	Boric acid H_3BO_3	Flowthrough	Reconstituted water; hardness 200 mg $CaCO_3$/liter	9-day NOEC-LOEC	0.75-1.0	Birge and Black (1977)
Channel catfish (*I. punctatus*), embryo-larval stages	Borax $Na_2B_4O_7 \cdot 10\ H_2O$	Flowthrough	Reconstituted water; hardness 50 mg $CaCO_3$/liter	9-day NOEC-LOEC	9.0-25.9	Birge and Black (1977)

TABLE 6
Summary of Boron Toxicity Data for Fresh Water Fish

Test organism	Boron compared tested	Type of test	Water quality characteristics	Test response(s) reported	Boron concentration (mg B/liter)	Reference
Channel catfish (*I. punctatus*), embryo-larval stages	Borax $Na_2B_4O_7 \cdot 10\ H_2O$	Flowthrough	Reconstituted water; hardness 200 mg $CaCO_3$/liter	9-day NOEC-LOEC	0.49-1.04	Birge and Black (1977)
Goldfish (*Carassius auratus*), embryo-larval stages	Boric acid H_3BO_3	Flowthrough	Reconstituted water; hardness 50 mg $CaCO_3$/liter	7-day NOEC-LOEC	9.2-22.5	Birge and Black (1977)
Goldfish (*C. auratus*), embryo-larval stages	Boric acid H_3BO_3	Flowthrough	Reconstituted water; hardness 200 mg $CaCO_3$/liter	7-day NOEC-LOEC	6.8-8.33	Birge and Black (1977)
Goldfish (*C. auratus*), embryo-larval stages	Borax $Na_2B_4O_7 \cdot 10\ H_2O$	Flowthrough	Reconstituted water; hardness 50 mg $CaCO_3$/liter	7-day NOEC-LOEC	26.50-48.75	Birge and Black (1977)
Goldfish (*C. auratus*), embryo-larval stages	Borax $Na_2B_4O_7 \cdot 10\ H_2O$	Flowthrough	Reconstituted water; hardness 200 mg $CaCO_3$/liter	7-day NOEC-LOEC	8.53-27.33	Birge and Black (1977)
Rainbow trout (*S. gairdneri*), embryo-larval stages	Boric acid H_3BO_3	Flowthrough	Reconstituted water; hardness 50 mg $CaCO_3$/liter	28-day NOEC-LOEC	0.11-1.00	Birge and Black (1977)
Rainbow trout (*S. gairdneri*), embryo-larval stages	Boric acid H_3BO_3	Flowthrough	Reconstituted water; hardness 200 mg $CaCO_3$/liter	28-day NOEC-LOEC	0.001-0.01	Birge and Black (1977)

TABLE 6
Summary of Boron Toxicity Data for Fresh Water Fish

Test organism	Boron compared tested	Type of test	Water quality characteristics	Test response(s) reported	Boron concentration (mg B/liter)	Reference
Rainbow trout (*S. gairdneri*), embryo-larval stages	Borax $Na_2B_4O_7 \cdot 10\ H_2O$	Flowthrough	Reconstituted water; hardness 50 mg $CaCO_3$/liter	28-day NOEC-LOEC	0.96-9.70	Birge and Black (1977)
Rainbow trout (*S. gairdneri*), embryo-larval stages	Borax $Na_2B_4O_7 \cdot 10\ H_2O$	Flowthrough	Reconstituted water; hardness 200 mg $CaCO_3$/liter	28-day NOEC-LOEC	9.63-49.70	Birge and Black (1977)
Large mouth bass (*Micropterus salmoides*), freshly fertilized eggs	Boric acid H_3BO_3	Flowthrough	Reconstituted hard water 200 mg $CaCO_3$/liter	11-day NOEC-LOEC	1.39-12.17	Birge and Black (1981)
Rainbow trout (*S. gairdneri*), freshly fertilized eggs	Boric acid H_3BO_3	Flowthrough	Reconstituted hard water 200 mg $CaCO_3$/liter	32-day NOEC-LOEC	0.01-0.1	Birge and Black (1981)
Rainbow trout (*S. gairdneri*), early life stages	Boric acid H_3BO_3	Flowthrough	Natural water exposures	36-day NOEC-LOEC	0.75-1.0	Procter and Gamble (unpublished)
Rainbow trout (*S. gairdneri*), early life stages	Boric acid H_3BO_3	Flowthrough	Well water; water hardness 27 mg $CaCO_3$/liter; pH 6.5-7.5	60-day LOEC	>17	Procter and Gamble (unpublished)

[1]TLM = Median tolerance limit, the concentration of material in water at which 50% of the test organisms survive after a specified time of exposure.
Source: Butterwick et al. 1989

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